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Heart failure management: The importance of sleep-related breathing disorders

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Sleep-disordered breathing (SDB) results in an increased risk of hypertension, type 2 diabetes mellitus, coronary artery disease, all of which can predispose to heart failure (HF). The two types of HF – reduced ejection fraction (HFrEF) and preserved ejection fraction (HFpEF) – can both be affected by the presence of SDB. Increased arousals from sleep observed at the end of apneas cause an increase in sympathetic activity, elevating blood pressure. Intermittent hypoxia results in hypoxic vasoconstriction of pulmonary vessels, resulting in increased pulmonary artery pressures, and systemic inflammation. These are potential mechanisms by which SDB can worsen HF. Conversely, the prolonged circulatory times, increase in total body fluid retention, and respiratory control system instability can all contribute to SDB – both obstructive and central sleep apnea. Thus, a bidirectional relationship between SDB and HF exists and calls for the integration of SDB treatment in HF management. Treatment of SDB can mitigate the detrimental cardiovascular effects of the disease and contribute to the optimization of chronic HF management. Optimal treatment of SDB can result in systemic blood pressure reduction, improved control of atrial fibrillation recurrence, reduced pulmonary artery pressures, improvement in low inotropic states, and potentially improved effectiveness of diuresis. These effects can have clinical consequences of reduced hospitalizations and re-admissions for heart failure.

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